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remained uninfluenced by the changing rate of left ventricular filling.

In summary, the systolic and diastolic time intervals do not allow conclusions to be drawn as to the underlying mechanism responsible for the alternating contractions in patients with pulsus alternans. Spodick is right that our observations were made in patients with servere aortic valve disease, and should, therefore, be restricted to this group of patients.

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Transient Entrainment and Interruption of Ventricular Tachycardia With Rapid Atrial Pacing—I

In the report by Waldo, et al. (1), one minor point of interpretation was confusing. The authors suggested that the area of slow conduction in the ventricle was probably functionally present only during ventricular tachycardia because of the shortening of the stimulus to right ventricular electrogram interval by 335 ms immediately after interruption of the tachycardia and because of the return of the QRS morphology to the "normal" right bundle branch block pattern.

Several observations from the figures in their report in combination suggest that the opposite conclusion—that is, the continued presence of the slow conduction—would be the more likely: 1) the tachycardia has a left bundle branch block morphology, 2) entrainment at a rate of 165 beats/min resulted in a narrow QRS complex with relatively normal morphology and, most importantly, 3) the first paced beat after the interruption of the ventricular tachycardia showed a right bundle branch block morphology.

These three observations suggest that the right bundle branch was the area of conduction delay. The electrical circus movement thus conducted slowly down the right bundle branch in an anterograde direction, returned retrograde via the left bundle branch and reentered the right bundle. This would yield a left bundle branch block morphology to the ventricular tachycardia. During entrainment, the atrial paced beats conducted down both bundle branches, although more slowly down the right bundle branch. With increasing pacing rates, there was increasing fusion from the paced beat conducting down the left bundle branch and the previous paced beat conducting slowly down the right bundle branch. At termination of ventricular tachycardia, this fusion phenomenon ceased; that is, there was no longer any ventricular depolarization occurring via the right bundle branch from the antecedent paced beat, resulting in the right bundle branch block morphology of the "normal" QRS. This strongly suggests that the right bundle branch was the area of slow conduction. Were the right bundle branch not involved, one would not expect this sudden widening of the QRS morphology in association with termination of the reentrant loop.

That the stimulus to right ventricular electrogram interval decreased by 335 ms is entirely consistent with the fact that the right ventricle is now depolarized via the faster conducting left bundle branch. It need not imply that slow conduction has ceased over the right bundle branch. The suddenness of the termination of the ventricular tachycardia also need not imply that the slow conduction has ceased, only that an exit block is now present. One blocked conduction down the right bundle branch was all that was needed to allow retrograde penetration of the right bundle branch via the left bundle branch, resulting in persistence of the exit block and preventing reinitiation of the tachycardia after cessation of pacing.

One can also postulate that initiation of ventricular tachycardia can be easily achieved by a single ventricular premature beat. A properly timed ventricular premature beat can find the right bundle branch still refractory, but conduct retrograde up the left bundle branch. This could 1) allow it to enter the right bundle branch and set up the circus movement, or 2) block a sinus beat from conducting down the left bundle branch, thus allowing conduction through the right bundle setting up the circus pathway. It would seem that this case report was an excellent example of sustained ventricular tachycardia due to bundle branch reentry.

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Reference

1. Waldo AL, Henthorn RW, Plumb VJ, MacLean WAH. Demonstration of the mechanism of transient entrainment and interruption of ventricular tachycardia with rapid atrial pacing. J Am Coll Cardiol 1984;3:422–30.

Reply

We suggested that the area of slow conduction in the ventricles was only functionally present during ventricular tachycardia. Tchou concludes that the opposite is more likely. Actually, independent of whether we agree with Tchou's conclusion, we believe his point really is semantic. Perhaps it would have been better to have said "functionally operative" instead of "functionally present." Our point really is that the area of slow conduction is not demonstrable except during the period of the tachycardia and, if we understand Tchou's letter, we believe he would accept that.

Tchou's suggestion that the ventricular tachycardia represents a case of bundle branch block reentry is certainly intriguing. However, we would prefer not to speculate beyond that modest statement for several reasons. First, bundle branch block reentry as a cause of sustained ventricular tachycardia is very rare, if indeed

it ever occurs. Second, no mapping of the ventricles was performed to provide any data which could be used to analyze this possibility. Third, it is really not relevant to the points of the report regarding transient entrainment and interruption of a tachycardia with rapid pacing.

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Transient Entrainment and Interruption of Ventricular Tachycardia With Rapid Atrial Pacing—II

We read with great interest the report by Waldo et al. and the accompanying editorial about entrainment of ventricular tachycardia (1,2). Several additional points about this phenomenon merit emphasis because of their importance to the mechanisms of ventricular tachycardia. We agree with Brugada and Wellens (2) that entrainment per se does not imply reentry. However, if focal impulse formation were hypothesized to be the mechanism responsible for an entrainable ventricular tachycardia with fusion, a sector of unidirectional block out of the focus would have to be present for the entraining wave to access the site of abnormal impulse formation and accelerate the next beat. Reentry with an excitable gap, as indicated by Waldo and his colleagues, is a simpler and better explanation for ventricular tachycardia in the patient they describe.

More can be said about the reentry mechanism in their case. The point or points at which the wave of excitation from the reentrant circuit engages the rest of the myocardium must be separate from the path over which the entraining wave front gains access to the circuit, so that access is not blocked by the wave front that just emerged from the circuit. Furthermore, a large area of physiologic or anatomic block between the muscle depolarized by the entraining stimulus and that depolarized by the wave front emerging from the site of reentry is required for fusion to be manifest during entrainment. If this were not the case, a contribution to the activation sequence by the entrained impulse would be inapparent.

The simplest explanation for the findings of Waldo et al. is reentry around a large anatomic or physiologic barrier, or macroreentry. This mechanism would provide both for facile engagement of the reentry circuit by an entraining impulse and for fusion. We believe that ventricular tachycardia subject to entrainment with fusion constitutes strong evidence of macroreentry (3). Other explanations for ventricular tachycardia of this nature are contrived and complicated. Finally, the occurrence of entrainment of ventricular tachycardia proves that not all ventricular tachycardia is caused by "protected localized reentry" (4).

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2. Brugada P, Wellens HJJ. Entrainment as an electrophysiologic phenomenon. J Am Coll Cardiol 1984;3:451–4.
3. Anderson KP, Swerdlow CD, Mason JW. Entrainment of ventricular tachycardia. Am J Cardiol 1984;53:335–40.
4. Josephson ME, Horowitz LN, Farshidi A, Spielman SR, Michelson EL, Greenspan AM. Sustained ventricular tachycardia: evidence for protected localized reentry. Am J Cardiol 1978;42:416–24.

Reply

Waldo et al. have emphasized that transient entrainment can only be established by fulfilling one of three criteria: 1) constant fusion beats during rapid pacing at a constant rate except for the last captured beat; 2) progressive fusion (constant fusion beats at different rapid pacing rates but different degrees of fusion at the different rate); and 3) interruption of the tachycardia by rapid pacing associated with localized conduction block to a site followed by activation of that site from a different direction and with a shorter conduction time by the next pacing impulse. If one of these criteria can be fulfilled, Waldo et al. (1,2) have suggested this is best explained by reentry. Similarly, Brugada and Wellens (3) stated that "unless otherwise proved, demonstration of transient entrainment of tachycardia using the criteria of Waldo et al. is a very easy way to demonstrate that reentry is the underlying mechanism of the arrhythmia" (3). Thus, both Waldo et al. (1,2) and Brugada and Wellens (3) emphasize the point that one must be able to demonstrate one of the proposed criteria in order to demonstrate transient entrainment and, therefore, reentry. Furthermore, they have emphasized that with available data, only reentry can satisfactorily explain the observations that fulfill any of the three proposed criteria.

We agree it is likely that ". . . a large area of physiologic or anatomic block between the muscle depolarized by the entraining stimulus and that depolarized by the wave front emerging from the site of reentry is required for fusion to be manifest during entrainment." However, we do not understand clearly their statement that "the point or points at which the wave of excitation from the reentrant circuit engages the rest of the myocardium must be separate from the path over which the entraining wave front gains access to the circuit, so that access is not blocked by the wave front that just emerged from the circuit." The point is that to obtain transient entrainment, there must be an excitable gap in the reentrant circuit. The wave front from the pacing impulse which